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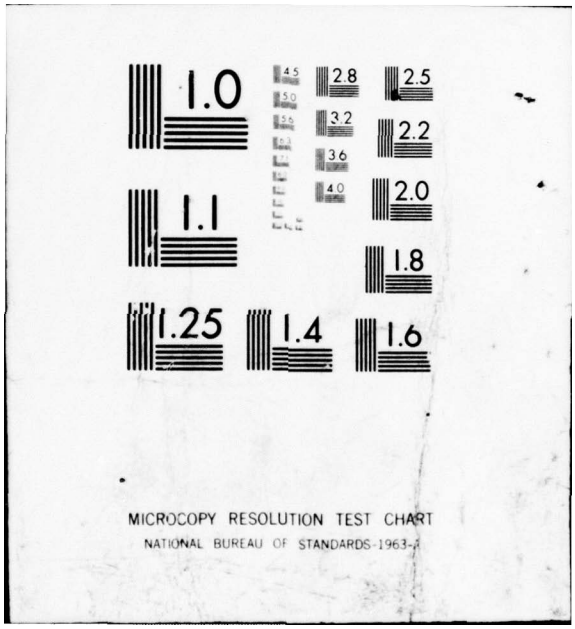
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MECHANISMS OF THE CHANGES IN ARTERIAL OXYGEN SATURATION AT ALTITUDE*

by

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The opinions or assertions contained herein are the private views of the authors and are not to be construed as official or as reflecting the views of the Department of the Army or the Department of Defense.

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The ratio of arterial oxygen content to its capacity is called arterial oxygen saturation (SaO_2). Its level is dependent on the alveolar gas exchange efficiency. A normal person, breathing room air at sea level, has an average of 97% SaO_2. This saturation may fall in a variety of conditions, such as low oxygen breathing or pulmonary disease. It is well known that blood oxygen saturation bears a relationship with the partial pressure of oxygen to which it is exposed. This relationship, known as the oxygen-hemoglobin (O_2-Hb) dissociation curve, is also modified by several factors, i.e., pH, temperature.

When a man goes to altitude, the arterial oxygen saturation is modified for many reasons. First, because of the low barometric pressure, the inspired oxygen tension (P_IO_2) is low. Therefore the alveolar and the arterial oxygen tension (PaO_2) are lower than sea level values. In short, the magnitude of the SaO_2 will depend on the P_IO_2. Second, once the PaO_2 has decreased, the arterial chemoreceptors will be stimulated, therefore the ventilation will be increased. Hyperventilation influences the SaO_2 by two mechanisms (fig. 1): a) increasing the PaO_2, which will increase SaO_2 according to the O_2-Hb dissociation curve relationship, and b) producing respiratory alkalosis, which will affect the O_2-Hb dissociation curve, shifting it to the left through the pH effect. Average values of SaO_2, pH and PaO_2 obtained in four sea level subjects (3,10) which sojourned to 4350 m (Cerro de Pasco, P_B 461 torr) are
1 depicted in fig. 1. The circle containing a cross represents two days, and the circle with an X indicates ten days at altitude. On the assumption these subjects would not have hyperventilated, their arterial blood could be predicted to be 32 torr P_{O_2}, 62% S_{O_2}. Starting from this point, the dotted line shows the increase of SaO_2 to 83% due only to increase in PaO_2 (constant pH).

The interrupted line labeled alkalosis shows the additional increase in SaO_2 to 88% due to the pH effect.

Third. Several investigators have shown that high altitude exposure produces a rightward shift of the O_2-Hb dissociation curve (1,6-8). P_{50} (7.4) determinations made on the same subjects (4), are shown in the lower part of fig. 1. Average values were 24 and 28 torr at sea level and high altitude, respectively. The saturation marked on the ordinate should not be considered in this case, since P_{50} by definition is the P_{O_2} pressure at 50% saturation. However, that ordinate gives an idea of the magnitude of the saturation changes that O_2-Hb dissociation curve shift will produce in the 24-28 torr range. Although the rightward shift of the O_2-Hb dissociation curve at altitude would facilitate the O_2 release at tissue level, it is seen in fig. 1 that P_{50} measurements obtained at sea level and at altitude, when calculated to the in vivo (full symbols) pH, have almost the same value. This means that, the shift described in vitro does not have any effect in vivo. Indeed, SaO_2 measured was 89% and not 82% that would be obtained were the in vitro phenomenon to take place. Similar conclusion has been reported previously (8).

Once the SaO_2 has been reached at altitude in the resting condition, it is further modified when a person performs exercise. Fig. 2 shows the changes in SaO_2 observed in the same subjects. Average values obtained in 8 high altitude natives are also included (3,9). This observation, i.e., the fall in SaO_2 with exercise at altitude, has been published by several investigators (2,5,11,12). The mechanisms that produce this SaO_2 fall are discussed under the remaining three points.

Fourth. Once again, SaO_2 will be modified by the ventilation. However the increase in ventilation obtained during exercise is adequate for the metabolic demands, except during heavy work. The final PaO_2 obtained during exercise will therefore depend on the alveolar gas efficiency. Our subjects
3 did not modify their PaO_2 during heavy work (fig. 3). Thus the SaO_2 fall observed, is fully explained by the metabolic acidosis (interrupted line labeled acidosis). However, additional factors are taking place that further change the SaO_2.

Fifth. An increase in body temperature occurs during exercise. Assuming a 2°C increase, the calculated saturation would fall to 79% (dotted line of fig. 3) largely because the pH becomes more acid, due to the temperature effect on pH. However at the same time the P_{O_2} rises due to the temperature effect on P_{O_2} (vertical dotted line of fig. 3) and the final calculated saturation is 86%.

Sixth. We have found that during heavy exercise, P_{50} (7.4) shifted to the left (4). Average values are shown in the lowest part of figure 3 at rest (R) and during heavy work (E). The in vivo P_{50} at rest and during exercise are also shown (full symbols). Taking this mechanism into account, i.e., left shift of the O_2-Hb dissociation curve, the SaO_2 would further rise to 89%.

It is concluded that it appears possible that SaO_2 does not change at all in vivo, and what we observe are the in vitro changes due to the acid pH. The same mechanisms in the discussion applies to high altitude native subjects since no differences are observed in SaO_2 when compared with sea level subjects (fig. 2).

Summary

The reasons for the arterial oxygen saturation (SaO_2) changes during altitude exposure at rest and during exercise are presented and discussed. SaO_2 is prevented to show lower values than usually measured due to hyperventilation. Ventilation increases SaO_2 through an elevation of P_{O_2} and pH. No negative contribution is found with the rightward shift of the O_2-Hb dissociation curve reported in vitro. The explanation is found on similar P_{50} *in vivo* values shown at sea level and at altitude. The SaO_2 fall observed during exercise at altitude is fully explained by the metabolic acidosis (Bohr effect). However, if additional factors are taken into account, such as temperature increase and left shift of the O_2-Hb dissociation curve, no changes are expected to occur in vivo.

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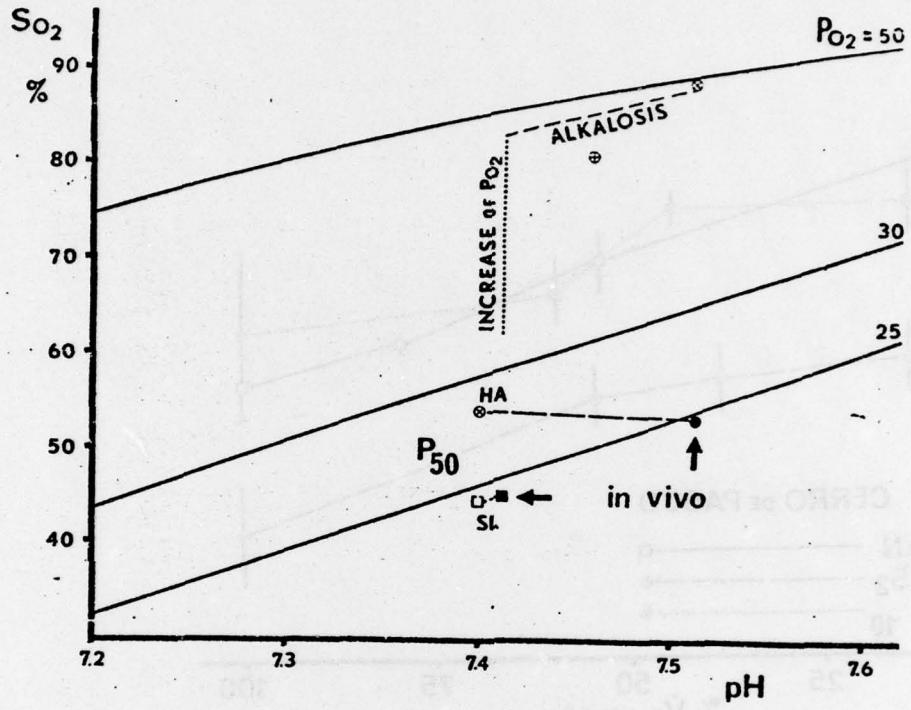
FIGURE LEGENDS

Fig. 1 Arterial oxygen saturation (SaO_2) changes due to altitude exposure in resting conditions. Mean values of SaO_2, pH and PaO_2 obtained in four sea level subjects sojourning at 4350 m are plotted on a saturation-pH diagram. Circle with a cross: 2nd day, and circle with an X: 10th day at altitude. Three iso-PO_2 lines are also drawn. The changes in SaO_2 due to hyperventilation (62 to 88%) are represented a) by an increase of PO_2 at a constant pH (dotted line) and b) by the resultant alkalosis at a constant PO_2 (dashed line). Mean P_{50} values obtained at sea level (SL, circles) and at altitude (HA, squares) are also shown. Saturation scale should not be considered in this case. The calculated in vivo P_{50}'s (full symbols) show similar values at SL and at HA, which explain the lack of effect of the rightward shift of the O_2-Hb dissociation curve on SaO_2.

Fig. 2 Mean $\pm$ SE of arterial oxygen saturation (SaO_2) as a function of relative oxygen uptake obtained at high altitude, 4350 m (9,10). Note the low SaO_2 values obtained at the 2nd day at altitude (dashed line) of sea level subjects (SLS) and their increase at the 10th day (dotted line) reaching similar values to high altitude natives (HAN, continuous line).

Fig. 3 Arterial oxygen saturation (SaO_2) changes obtained with heavy exercise at altitude. Similar diagram to figure 1. Symbols are mean values obtained at rest (R, circles) and during maximum oxygen uptake (E, squares) in four sea level subjects after 10 days at altitude. The arterial oxygen saturation (SaO_2) change observed is fully explained by the pH shift due to

metabolic acidosis (dashed line labeled acidosis). However, if we take into account the effect of temperature effect on pH and PO_2 (dotted line) and the left shift of P_{50}, 7.4 (continuous line), the final SaO_2 is similar to the resting value, which suggests the possibility that no changes in SaO_2 are taking place in vivo. Mean P_{50} values shown in the lower part of the figure as discussed in fig. 1.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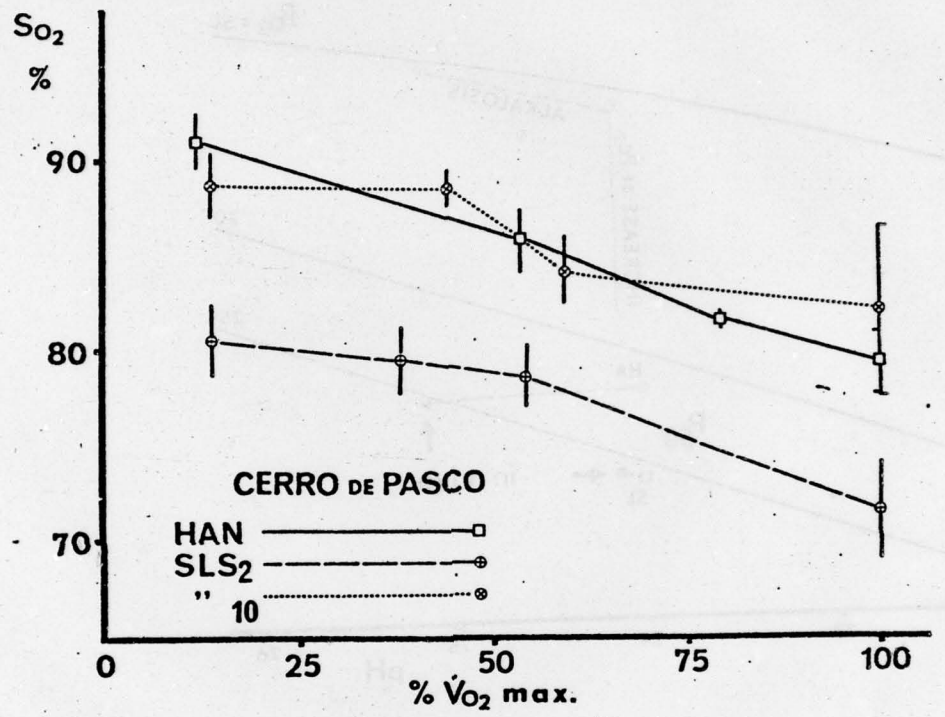
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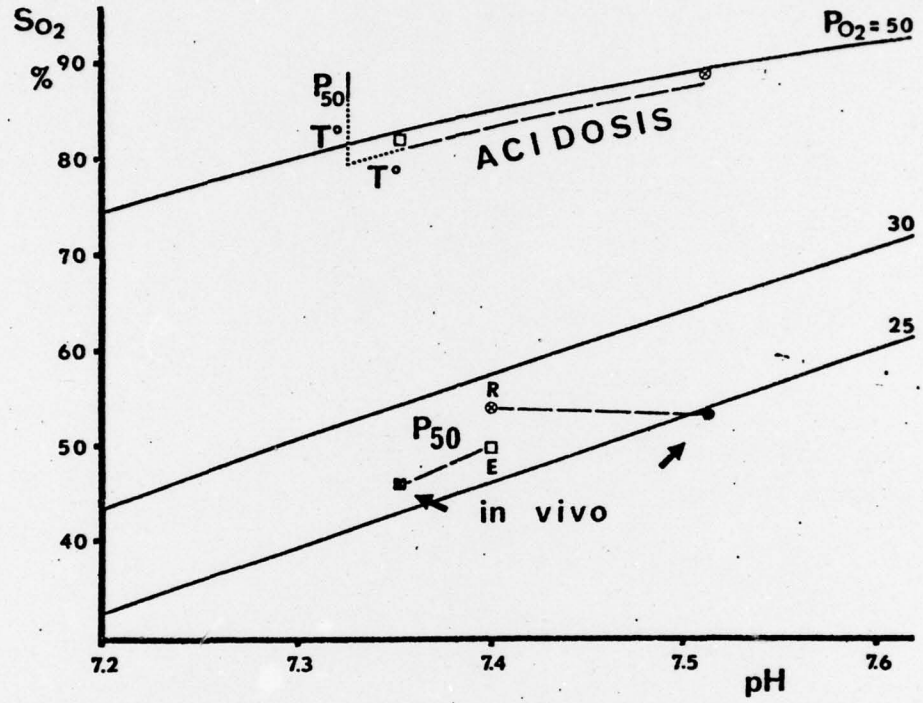
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P sub O_2

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The reasons for the arterial oxygen saturation (SaO_2) changes during altitude exposure at rest and during exercise are presented and discussed. SaO_2 is prevented to show lower values than usually measured due to hyperventilation. Ventilation increases SaO_2 through an elevation of P_{O_2} and pH. No negative contribution is found with the rightward shift of the O_2-Hb dissociation curve reported in vitro. The explanation is found on similar P_{50} in vivo values shown at sea level and at altitude. The SaO_2 fall observed during exercise at (cont next pg)

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